

L10 ANSWER 34 OF 34 CAPLUS COPYRIGHT 2002 ACS

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TITLE: **Production** of insulin-like growth factor-II (MSA) by endoderm-like cells derived from embryonal carcinoma cells: possible mediator of embryonic cell growth

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CORPORATE SOURCE: Lab. Tumor Immunol. Biol., Natl. Cancer Inst., Bethesda, MD, 20205, USA

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AB The present study was carried out to det. if an insulin-like growth factor

(IGF) type activity might be produced by embryonal carcinoma-derived cells. The cell line used to condition growth medium for the isolation of

secreted growth factors was a newly established Dif 5 cell type. Dif 5 cells are a differentiated endoderm-like cell type derived from F9 embryonal carcinoma cells (which possess properties similar to **mouse** embryonic stem cells) following extensive exposure to retinoic acid. When growth medium conditioned by Dif 5 cells was chromatographed on Sephadex G-75 in 1 M AcOH, 2 peaks of activity were obsd. which compete for specific 125I-labeled multiplication stimulating activity (MSA) binding to PYS cells. MSA is the rat **homolog** of **human** insulin-like growth factor II (IGF-II) [67763-97-7]. The high-mol.-wt. fraction [Mr .apprx.60 kilodalton (K)] apparently corresponds to IGF-binding **protein** as detd. by its ability to bind [125I]iodo-MSA. The low-mol.-wt. fraction (Mr .apprx.8K) is biol. activem, as this fraction stimulates [3H]thymidine incorporation into serum-starved chick embryo fibroblasts. RIA data indicate that the IGF-like activity produced by Dif 5 cells is more closely related to IGF-II than to IGF-I. Undifferentiated embryonal carcinoma stem cell lines (F9, Nulli, and PCC4) produced little of this MSA-like activity, whereas PYS-2 (parietal endoderm-like) cells produced about 16 ng MSA/106 cells/24 h as detd. by RIA. Dif 5 and PSA-5E (visceral endoderm-like) cells secreted significant amts. of MSA into the growth medium (30-50 ng MSA/106 cells/24 h). These findings offer further support to a proposal that MSA (IGF-II) produced by endoderm cells, particularly visceral endoderm, may serve as an early embryonic growth factor.

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MEDLINE

DUPLICATE 11

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TITLE: Adipsin and an endogenous pathway of complement from
adipose cells.
AUTHOR: Choy L N; Rosen B S; Spiegelman B M
CORPORATE SOURCE: Dana-Farber Cancer Institute, Harvard Medical School,
Boston, Massachusetts 02115.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1992 Jun 25)
267 (18) 12736-41.
Journal code: HIV; 2985121R. ISSN: 0021-9258. *unref.*
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AB The alternative complement pathway is best known for its role in humoral suppression of infectious agents. We have previously shown that adipose cells synthesize adipsin, the **mouse homolog** of **human** complement factor D, and that the synthesis of this **protein** is reduced in several rodent models of obesity. We show here that adipose cells and adipose tissue also synthesize two other essential components of the alternative pathway of complement, factors C3 and B, and activate the proximal portion of this pathway. This activation occurs in the absence of infectious agents and without triggering the terminal, lytic part of this pathway. We demonstrate the **production** in vitro of several polypeptides characteristic of complement activation that are known to have potent biological activities, including the anaphylatoxin C3a. Cultured adipocytes require stimulation with cytokines to activate complement, while explanted adipose tissue has no such requirement. The adipose tissue from obese mice is deficient in this localized activation of the alternative pathway. These results indicate that complement activation occurs in a localized site, adipose tissue, in normal mice and is impaired in a state of metabolic dysfunction. This suggests a novel function for the proximal portion of this complement pathway related to adipose cell biology or energy balance.